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Dental Indicators of Stress and Reduced Age at Death in Prehistoric Native Americans

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ABSTRACT Considerable evidence supports the hypothesis that developmental enamel defects represent stress-induced growth disruptions. In this investigation, the relationship between different kinds of enamel defects and age at death is examined in the prehistoric Libben population from Ottawa County, Ohio. The sample consisted of the permanent dentitions of 143 individuals. Defects were classified based on the criteria of the Developmental Defects of Enamel (DDE) Index. The multifactorial age at death determinations of Lovejoy and coworkers (1977) were used in this analysis. Results reveal a significantly lower mean age at death for individuals with enamel defects vs. individuals with normal teeth. This pattern was clearly present for all defect types examined. No significant differences by sex were detected. The age-at-death distribution for individuals with normal teeth approximated the normal curve. The modal value was reached in the 35–40 year age class. The age-at-death distribution for individuals with enamel defects showed two peaks. The mode occurred in the 15–20 year age class, and the second, lower peak occurred in the 30–35 year age class. The early mortality of individuals with enamel defects may be related to biological damage to the immune system during prenatal or postnatal development.

The evaluation of the health status of prehistoric populations has long been of major concern to biological anthropologists. These populations provide natural laboratories for testing hypotheses concerning the evolution and ecology of human disease. Indicators of general metabolic stress represent an important source of data for the reconstruction of prehistoric health status. Stress has been defined as a stereotypic physiological reaction to environmental insult (Goodman et al., 1988). It involves the activation of the pituitary–adrenal cortical axis and the sympathetic–adrenal medullary axis with increased release of adrenal cortical and adrenal medullary hormones (Goodman et al., 1980; McCance, 1990; Selye, 1971). One result of severe stress is growth disruption. Previous studies have demonstrated that bones and teeth are particularly susceptible to stress-induced growth disruption (Acheson, 1960; Park, 1964; Suckling and Thurley, 1984). Skeletal and dental indicators of stress are nonspecific in nature and include radiographic lines of increased density (Harris lines), fluctuating dental asymmetry, microscopic developmental enamel defects (Wilson bands), and macroscopic developmental enamel defects (hypoplasias and hypocalcifications).

In recent years, considerable attention has been focused on enamel hypoplasias, and to a lesser extent hypocalcifications, as indicators of early childhood stress. Numerous

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clinical, epidemiological, and experimental studies have demonstrated that developmental enamel defects represent stress-induced growth disruptions. Through clinical and epidemiological studies, enamel hypoplasias and hypocalcifications have been shown to be associated with a wide variety of systemic diseases, neonatal disturbances, and nutritional deficiencies in humans. These include: vitamin A deficiency (Wolbach, 1947), vitamin D deficiency (Nikiforuk and Fraser, 1981), premature birth (Rosenzweig end Sahar,1962), maternal diabetes (Noren, 1984; Grahnen and Edlund, 1967), neonatal asphyxia (Grahnen et al., 1969), neonatal jaundice (Watson et al., 1964), nephrotic syndrome (Schusterman and Fellers, 1969), gastroenteritis (Smith and Miller, 1979), hemolytic anemia (Pindborg, 1970), allergy (Rattner end Meyers, 1962), infectious diseases (Pindborg, 1982), neurological disturbances (Bhat and Nelson, 1989; Cohen and Diner, 1970), protein-calorie malnutrition (Baume and Meyer, 1966; Sawyer and Nwoku, 1985), nonspecific childhood diarrhea (Lindemann, 1958), and abnormal gestational events (Via and Churchill, 1959).

Experimental studies were undertaken during the period from 1940 to 1960 by Kreshover and coworkers. These studies clearly demonstrated that enamel defects could be produced experimentally using a wide variety of stressors including tuberculosis in mice (Kreshover, 1942), artificially induced fever in rats (Kreshover and Clough, 1953), alloxan diabetes in rats (Kreshover et al., 1953), and vaccinia infection in rabbits (Kreshover et al., 1954). Kreshover, in reviewing the possible causative factors in developmental tooth defects, states:

> abnormal tooth formation is a generally nonspecific phenomenon and can be related to a variety of local or systemic disturbances, any of which, depending upon their severity and the degree of tissue response, may result in defective enamel and dentin, either of the so-called hypoplastic or hypocalcified variety (1960: 161).

More recently, Suckling and coworkers have undertaken experimental studies using sheep. They have produced enamel defects experimentally in these animals through physical trauma (Suckling, 1980), fluorosis (Suckling and Thurley, 1984), and induced parasitism (Suckling et al., 1986).

In this study, the relationship between different kinds of enamel defects and age at death will be examined, to test the hypothesis that individuals who are stressed early in life suffer a different pattern of mortality than those who are not. There are several possible outcomes to this analysis:

1. A lower mean age at death for individuals with defects would suggest either:
   A) individuals with enamel defects represent socially disadvantaged individuals who face a lifelong pattern of culturally determined increased exposure to environmental stressors, resulting in enamel defects in childhood and early mortality in adulthood;
   B) individuals exposed to stress prenatally or in early childhood are biologically "damaged" by the stress and therefore have a reduced ability to cope with later insults;
   C) individuals with a genetic susceptibility to certain types of stressors may show a lifelong pattern of illness due to those stressors resulting in hypoplasias in childhood, and early mortality in adulthood; or
   D) in a burial site which spans many years, individuals with enamel defects may represent persons who lived during periods of resource scarcity. Both the enamel defects and the early mortality would result from the resource scarcity. (A, B, and C modified from Goodman and Armelagos, 1988.)

It should be noted that these potential mechanisms of early mortality are not mutually exclusive.

2. No difference in mean age at death for individuals with and without defects would suggest that either:
   A) early childhood stress has no effect on mortality; or
   B) the defects examined in this population are not the result of early childhood stress.
3. A higher mean age at death for individuals with defects would suggest that

individuals exposed to certain highly prevalent stressors in early childhood acquire an immunity to those stressors which protects them in adulthood. This is considered the least likely of the three possible outcomes.

TABLE 1. Dental wear rating

0	No noticeable wear.
1	Polishing on cusps, thin linear exposure of dentin on incisors.
2	Point exposure of dentin on cusps, thicker linear exposure on incisors.
3	Broad facets on cusps, 20–30% crown loss.
4	Coalescence of cusps, 30–50% crown loss.
5	Fifty percent or greater loss of crown. Dentin exposed as a broad uninterrupted surface.

ARCHAEOLOGICAL AND DEMOGRAPHIC CONTEXT OF THE SAMPLE

The sample used in this study was derived from the Libben population, a Late Woodland skeletal series housed at Kent State University. The Libben site is located on the Portage River in Ottawa County, Ohio, and was excavated during 1967 and 1968 under the direction of Dr. Olaf H. Prufer. Excavation produced a well preserved skeletal sample of 1,327 individuals, representing the largest prehistoric population yet uncovered from a single site in North America. Archaeological evidence indicates a year-round occupation of the site for a time span of 250–300 years. The site has been radiocarbon dated to between 800 and 1100 A.D. Culturally, the site has been determined to represent a novel "Libben phase" of the Western Basin Tradition of southern Michigan. While a small number of intrusive refuse pits were found, the vast majority of cultural material is clearly referable to the Libben phase (Lovejoy et al., 1977).

Stable carbon isotope and trace element analyses are not currently available for the Libben site. The analysis of faunal and floral remains, however, suggests a diet rich in animal protein. The vertebrate faunal assemblage consisted of over 301,500 pieces of bone. Faunal analysis resulted in the identification of 25 species of mammals, 11 species of fish, and 28 species of birds. Fish were of particular importance, providing 78.3% of the total projected poundage of usable meat. Utilized species included freshwater drum, walleye, catfish, bullhead, bass, pike, and sucker. Mammals provided 20.2% of the total projected poundage of usable meat. Utilized species included white-tail deer, raccoon, muskrat, black bear, and elk. While numerous bird species were represented at the site, their individual frequencies were low. Birds appeared to be relatively unimportant for subsistence, providing only 1.4% of the total projected poundage of usable meat (Romain, 1979).

Few vegetable remains were found, although their scarcity is probably due to differential preservation (Harrison, 1978). Analysis of charred floral remains revealed the presence of 224 hickory nut shell fragments, 412 acorn shells, 82 corn kernels, as well as lesser quantities of raspberry, hackberry, and dock seeds (Romain, 1979). The corn kernels found at the site suggest the possibility of marginal maize agriculture at some time during the occupation.

Demographic analysis of the skeletal population indicates a mean life expectancy at birth of 20 years. Infant mortality was low relative to that expected based on the overall death rate. Among adults, male mortality was consistently higher than that of females (Lovejoy et al., 1977).

MATERIALS AND METHODS

Selection of the sample used in this study was not strictly random, as an attempt was made to fill 5 year age-at-death intervals. Selection of individuals within these 5 year intervals was, however, random. The multifactorial age assessment of Lovejoy et al. (1977) was accepted for purposes of analysis. Nonageable individuals were deleted from the sample, while nonsexable individuals for whom reliable ages were available were included. The degree of dental wear was assessed for each tooth using a six point scale, shown in Table 1. Teeth with dental wear ratings of 4 or 5 were deleted from the sample. In a study of the distribution of enamel hypoplasias within teeth, Goodman and Armelagos (1985) found that for all tooth types, hypoplasias were far more common in the middle and cervical thirds than in the occlusal third. Thus, loss of enamel near the

occlusal surface due to attrition is likely to be of minimal significance.

Teeth showing severe premortem damage due to caries or postmortem damage due to fracture were also deleted. At least six undamaged permanent teeth needed to be present for an individual to be included in the sample. At least three of these needed to come from the anterior dentition (incisors, canines). The latter was required due to the greater sensitivity of the anterior teeth to hypoplasia (Goodman and Armelagos, 1985). It should be noted that, while these criteria were chosen to establish a minimum requirement, most individuals included in the sample had considerably more complete dentitions. The culling process described here yielded a final sample of 143 individuals.

Each dentition was thoroughly cleaned with alcohol and examined using a stereomicroscope and magnifying lens. All enamel defects were initially classified based on the criteria of the Developmental Defects of Enamel (DDE) Index (Federation Dentaire International, 1982). Linear hypoplasias were graded for severity based on width, depth, and definition. They were scored as grade C (mild), B (moderate), or A (severe). Those classified as grade C were often questionable cases, consisting of perikymata which were only slightly broadened. Goodman and Rose have recently stated that:

> most enamel hypoplasias are associated with abnormal histological changes (accentuated stria of Retzius or "Wilson" and "Cluster" bands). However, the lack of association of some mild surface irregularities, characteristically seen as thin, perikymata-like surface depressions, with abnormal prism morphology suggest that these surface features may not be evidence of physiological perturbation (1990:59).

The grade C category used in this study is consistent with this assessment. Linear hypoplasias classified as grade B were well defined horizontal grooves of developmental origin which did not exceed 0.50 mm in width. Those classified as grade A were grooves which exceeded 0.50 mm in width or resulted in gross morphological changes such as enlargement of the buccal pit or deformation of cusps.

Pitting hypoplasias were classified as being present or absent. Hypocalcifications (opacities) were classified as "demarcated" if they possessed clearly defined boundaries and "diffuse" if they lacked clear boundaries, following the guidelines of the DDE Index.

TABLE 2. Mean age at death for individuals with enamel defects of any kind vs. those with normal teeth

	Normal individuals	Individuals with defects
Mean	32.29	26.92
SD	11.17	11.85
SE	1.35	1.38
N	69	74
Pooled t test t = −2.78	df = 141	2-tail prob. = 0.006

RESULTS

The mean age at death for individuals with enamel defects of any kind vs. those with normal teeth is shown in Table 2. Individuals with enamel defects had a mean age at death which was 5.37 years lower than individuals with normal teeth. This result was significant at the 0.01 level based on the pooled t-test.

Table 3 shows the mean age at death for individuals with different kinds of defects (analyzed separately) vs. those with normal teeth in the permanent dentition. Individuals with grade A or B linear hypoplasias died on average 5.88 years younger than individuals with normal teeth ($P < 0.004$). The mean age at death for individuals with pitting hypoplasias was 13.15 years lower than for individuals with normal teeth ($P < 0.004$). Individuals with demarcated hypocalcifications died 7.64 years younger ($P < 0.003$), and those with diffuse hypocalcifications 17.49 years younger ($P < 0.001$) than individuals with normal teeth.

Sex-specific analyses were restricted to linear hypoplasias, as these were the most frequently occurring defects. Table 4 shows the mean age at death for males and females, with and without grade A or B linear hypoplasias in the permanent dentition. Males with grade A or B linear hypoplasias had a mean age at death which was 5.02 years lower than for males with normal teeth. This result was significant at the 0.05 level. Females with grade A or B linear hypoplasias showed a mean age at death which was 7.14 years lower than for females with normal teeth. This result was also significant at the

TABLE 3. Mean age at death for individuals with different kinds of enamel defects (analyzed separately) vs. those with normal teeth

	Normal individuals	Individuals with grade A or B LHP	Individuals with PHP	Individuals with DemHC	Individuals with DifHC
Mean	32.29	26.41	19.14	24.65	14.80
SD	11.17	11.85	11.23	12.44	12.28
SE	1.35	1.52	4.25	2.24	5.49
N	69	61	7	31	5
t (pooled *t* test)		2.91	2.96	3.05	3.36
df		128	74	98	72
2-tail prob.		0.004	0.004	0.003	0.001

TABLE 4. Mean age at death for males and females, with and without grade A or B linear hypoplasias

	Normal males	Males with LHP	Normal females	Females with LHP
Mean	33.22	28.20	37.04	29.90
SD	7.35	9.71	10.82	10.86
SE	1.30	2.17	2.21	2.02
N	32	20	24	29
t (pooled *t* test)		2.11		2.39
df		50		51
2-tail prob.		0.039		0.021

TABLE 5. Two-way analysis of variance for males vs. females, with and without grade A or B linear hypoplasias

Source of variation	df	F	Significance
Main effects	2	5.56	0.005
Sex	1	2.20	0.141
LHP	1	10.16	0.002
2-way interactions	1	0.307	0.581
Sex/LHP	1	0.307	0.581
Explained	3	3.81	0.012
Residual	101		
Total	104		

0.05 level, and approached significance at the 0.01 level.

A two-way analysis of variance was run for males vs. females with and without linear hypoplasias, to test the hypothesis that the two sexes differed in their mortality response to the presence of linear hypoplasias. The results were not significant for sex, but did confirm the very strong relationship between the presence of linear hypoplasias and reduced age at death. The results of this analysis are shown in Table 5.

Figure 1 shows the age-at-death distribution for individuals with normal teeth. This distribution approximates the normal curve and shows minimal kurtosis. It is unimodal, and the modal value is reached in the 35–40 year age class. Figure 2 shows the age-at-death distribution for individuals with defects of any kind. This distribution shows considerable negative kurtosis. It has two peaks, with the mode occurring in the 15–20 year age class, and the second, lower peak occurring in the 30–35 year age class. The two distributions are significantly different based on the Kolmogorov–Smirnov two-sample test.

Kolmogorov–Smirnov two-sample tests were also used to test for differences between the age-at-death distributions of individuals with normal teeth and those with different kinds of defects analyzed separately. The results of this analysis are shown in Table 6. The distributions for individuals with linear hypoplasias, demarcated hypocalcifications, and diffuse hypocalcifications are each significantly different than that for normal teeth. No significant difference was detected for pitting hypoplasias.

DISCUSSION

The results of this analysis reveal a significantly lower mean age at death for individuals with enamel defects, when compared to individuals with normal teeth. This pattern was clearly present for all defect types examined. Significant differences were also found in the age-at-death distributions of individuals with enamel defects vs. those with normal teeth.

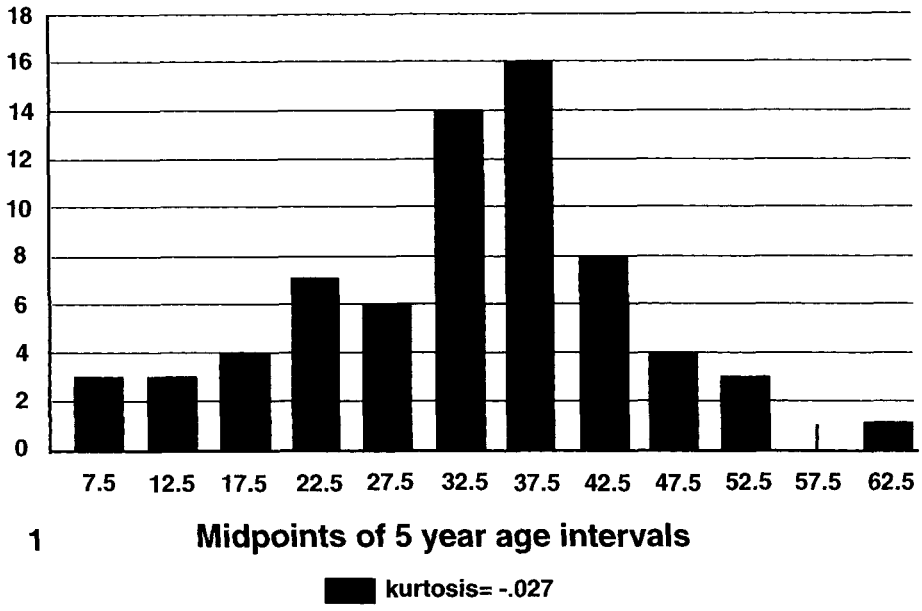

Fig. 1. Age-at-death distribution for individuals with normal teeth.

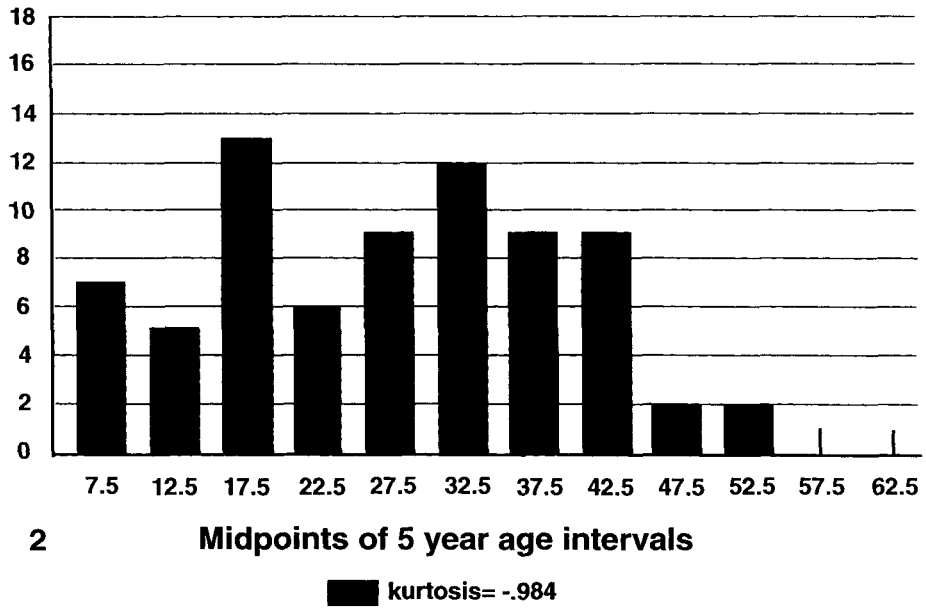

Fig. 2. Age-at-death distribution for individuals with enamel defects.

TABLE 6. Results of the Kolmogorov–Smirnov two-sample tests

Normal vs.	N	K-S Z	2-tail prob.
LHP	130	1.50	0.022
PHP	76	1.33	0.059
DemHC	100	1.70	0.006
DifHC	74	1.48	0.025

At least four previous studies have dealt with the relationship between enamel defects and age at death. In a study of enamel hypoplasias in South African Australopithecines, White (1978) found that the dentitions of individuals with hypoplasias were at an earlier stage of overall development than the dentitions of individuals without hypoplasias. Cook and Buikstra (1979) compared the mean age at death of subadults with and without enamel defects of the deciduous teeth, in Middle and Late Woodland samples from Illinois. They found that enamel defects were associated with decreased longevity in both samples.

Goodman and Armelagos (1988) examined the relationship between linear hypoplasias and age at death in the three different cultural horizons of the Dickson Mounds population. They found the relationship to be nonsignificant in the earliest, Late Woodland sample. In the Mississippian Acculturated Late Woodland sample, individuals with one hypoplasia/stress episode died 5.5 years younger than individuals with normal teeth, while individuals with two or more hypoplasia/stress episodes died 8 years younger than individuals with normal teeth. In the latest, Middle Mississippian sample, individuals with one hypoplasia/stress episode died 7.3 years younger than individuals with normal teeth, while individuals with two or more such episodes died 15.7 years younger than individuals with normal teeth. This pattern of increasing mortality of stressed individuals over time is concordant with other skeletal and dental indicators, which suggest a deterioration in the health status of the Dickson Mounds population during Middle Mississippian times (Lallo et al., 1978; Goodman et al., 1980).

Simpson and coworkers (1990) examined enamel hypoplasias and age at death in the Santa Catalina de Guale skeletal population from coastal Georgia. They found that fewer adults were affected by hypoplasias than subadults, although the differences were not statistically significant.

Studies of skeletal stress indicators other than enamel defects lend further support to the hypothesis that early childhood stress results in reduced age at death. In a study of tooth crown size and age at death in prehistoric Native Americans from the Averbuch site in Tennessee, Guagliardo (1982) found that the permanent teeth of juveniles were significantly smaller than those of adults. Archaeological and biological evidence suggests that the Averbuch population suffered considerably from environmental stressors. The findings of this study suggest that teeth may fail to develop to their maximum genetic size potential in the presence of chronic environmental stressors (Guagliardo, 1982). Clark and coworkers (1986) found small vertebral neural canal size to be associated with an earlier age at death in the Dickson Mounds population. This study is discussed further below.

The findings for Libben are in fundamental agreement with these earlier studies. The hypothesis that individuals who are stressed early in life suffer an earlier mean age at death appears to be strongly supported. The mechanism responsible for this early mortality of stressed individuals remains to be elucidated. The four mechanisms outlined in the introductory remarks will be discussed.

Individuals with enamel defects represent socially disadvantaged individuals who face a lifelong pattern of culturally determined increased exposure to environmental stressors, resulting in enamel defects in childhood and early mortality in adulthood

There are two possible ways in which this mechanism could operate. The first would involve the presence of clearly defined social classes with unequal access to vital resources. Such classes are reflected in the archaeological record by socially segregated burial sites and the differential presence of burial goods. At Libben, burial goods were not found to be restricted to a specific area of the site or type of burial (Prufer, personal communication). In a study of the distribution of

burial goods at Libben, Romain (1980) found no significant associations between age class or sex and the presence of burial goods, based on χ^2 tests. The archaeological evidence therefore does not support the existence of clearly defined social classes at Libben, and suggests an egalitarian social organization.

A second, more subtle way in which this mechanism might operate is through individually achieved status. Individuals who were proficient at hunting, gathering, or magicoreligious endeavors may have been better nourished, and better able to provide food for their families, than those who were not. This second alternative is more likely to be archaeologically invisible than the first, and may have played a role at Libben. It is unlikely, however, that this mechanism alone could account for the large number of individuals with enamel defects who suffered early mortality at Libben. If present, its role was probably secondary.

Individuals exposed to stress prenatally or in early childhood are biologically damaged by the stress, and therefore have a reduced ability to cope with later insults

The physiological stress response involves the activation of the pituitary–adrenal cortical axis and the sympathetic–adrenal medullary axis, with increased release of adrenal cortical hormones (corticosteroids) and adrenal medullary hormones (catecholamines). The stress response is considered generally adaptive in that it plays a major role in activating the body's defenses at the cellular, histological, organ, systemic, and organismal levels. If, however, stress becomes severe or prolonged, it can lead to suppression of the immune system and a variety of "diseases of adaptation (Selye, 1956)."

Suppression of the immune system has been shown to result directly from the action of corticosteroids (Claman, 1988). The adaptive role of corticosteroids in the stress response is to act as an immune system damper, preventing massive inflammation and mitigating against autoimmune disorders. The deleterious effects of corticosteroids on the immune system are profound and broad in scope. They inhibit production of the cytokines, including interleukin-l, interleukin-2, the IL-2 receptor, and interferon (Claman, 1988). They also reduce populations of eosinophils, lymphocytes, and macrophages, and suppress synthesis of immunoglobulin (McCance, 1990).

Prenatal exposure of individuals to corticosteroids alone has been shown to lead to a variety of neurological disturbances during subsequent development (Weichsel, 1977). These neurological symptoms are virtually identical to those observed in the offspring of female rats fed a diet high in ethanol alcohol during pregnancy (Taylor et al., 1988). This finding led Taylor and coworkers to hypothesize that the stress response itself might contribute to the specific effects of alcohol on the developing organism. They went on to examine the relationship between the alcohol-induced prenatal activation of the stress response and the subsequent ability of the organism to adapt to stress in the postnatal environment.

In these experiments, Taylor and coworkers (1988) found that the offspring of rats fed a diet high in ethanol showed significantly higher levels of brain and plasma corticosteroids at birth than did controls, reflecting prenatal activation of the stress response. These differences in corticosteroid levels quickly disappeared by the third day of life. The rats were allowed to mature to adulthood and were then exposed to a broad range of stressors including ethanol, cardiac puncture, shock, noise, shaking, and morphine injections. With each of these stressors, the prenatally ethanol-stressed rats showed a significantly greater rise in plasma corticosteroid levels than did controls. Responses to cold and fasting, however, did not differentiate between ethanol-stressed rats and controls (Taylor et al., 1988).

While more work is clearly needed using different prenatal and postnatal stressors, these experiments do suggest that prenatal exposure to stress can biologically alter an organism in such a way that subsequent exposure to postnatal stressors elicits an exaggerated corticosteroid response. Higher levels of circulating corticosteroids would lead to increased immune suppression, which would in turn lead to increased risk of infection and mortality.

Support for this hypothesis in the anthro-

pological literature is found in the work of Clark and coworkers (1986). These researchers examined the relationship between vertebral neural canal size and age at death in the Dickson Mounds population. The vertebral neural canal completes its growth at age 4 years, with subsequent growth of the vertebrae occurring in the vertebral body. These researchers found small vertebral neural canal size to be significantly associated with an earlier age at death. Vertebral neural canal size has the same growth curve as the thymolymphatic tissues. Small vertebral neural canal size may reflect a systemic perturbation occurring during prenatal or early postnatal development, resulting in damage to developing neural and thymolymphatic tissues, with consequent effects on neural and immune system function (Clark et al., 1986).

It is also possible that environmental stressors may biologically damage the immune system of the developing fetus or child through a mechanism other than the specific endocrine pathways of the stress response. Recent research suggests that iron deficiency during critical prenatal and postnatal periods can result in long-term impairment of immune system functioning which is not corrected by dietary iron supplementation after weaning (Sherman and Helyar, 1988). In a study of the offspring of rats fed an iron-deficient diet during gestation and lactation, Kochanowski and Sherman (1985) found a significant decrease in thymus cellularity and antibody response, which was not corrected by postweaning iron repletion.

Skeletal evidence suggests a high incidence of iron-deficiency anemia in Libben infants and children. In a study of the distribution of porotic hyperostosis and periosteal reactions in the subadults of the Libben population, Mensforth and coworkers (1978) found a high frequency of both lesions in individuals between 6 and 24 months of age. Porotic hyperostosis is a skeletal indicator of iron-deficiency anemia, while periosteal reactions are manifestations of infectious disease. The high frequency of iron-deficiency anemia at Libben does not appear to be due to a lack of dietary iron. As stated earlier, faunal and floral analysis of the site indicates a diet which included abundant animal protein. Such a diet would have been rich in bioavailable iron. It is more likely that the iron deficiency seen in Libben children between 6 and 24 months of age is the result of nutrient depletion due to a synergism between microbial infection, intestinal malabsorption, and increased nutritional demands accompanying a period of rapid somatic growth (Mensforth et al., 1978).

It appears likely that biological damage to the immune system played an important role in the early mortality of individuals with enamel defects at Libben. This biological damage may have occurred through prenatal activation of the stress response, iron deficiency during early childhood, or some interaction between the two.

Individuals with a genetic susceptibility to certain types of stressors may show a lifelong pattern of illness due to those stressors, resulting in enamel defects in childhood and early mortality in adulthood

Genetic explanations for disease and mortality patterns have always been attractive to physical anthropologists, as they offer a direct link to evolution. It would be logical to assume that individuals who, through genetically "weak constitutions," suffer chronic morbidity throughout life are also likely to suffer early mortality. Severe illness during childhood would be recorded on the teeth as enamel defects. Recurrent morbidity due to susceptibility to specific stressors would result in recurrent activation of the stress response. This in turn might initiate a positive feedback cycle in which increased corticosteroid secretion suppresses the immune system, resulting in increasingly violent bouts of illness, which in turn cause a further increase in corticosteroid secretion.

One problem with this mechanism concerns the degree to which resistance to disease is heritable. While a few balanced polymorphisms which convey resistance to specific diseases are known, numerous genetic studies have demonstrated that in general, traits which have a high correlation with fitness also show low heritability (Falconer, 1972). The differences in age at death between individuals with and without enamel defects at Libben are dramatic

rather than subtle. They occur during the prime reproductive years. This pattern is also seen in three of the four previous studies described above. Clearly, the traits which underlie these differences in age at death are highly correlated with fitness. A genetic explanation would therefore seem unlikely.

In a burial site which spans many years, individuals with enamel defects may represent persons who lived during periods of resource scarcity; both the enamel defects and the early mortality would result from the resource scarcity

During a 300 year time span, several periods of severe resource scarcity are almost certain to occur. Individuals of early childhood age alive during these periods of scarcity would be likely to experience nutritionally related morbidity. These episodes of morbidity would be recorded on the teeth as enamel defects. Malnutrition is known to interact synergistically with infectious disease, resulting in both severe morbidity and increased risk of mortality (Scrimshaw et al., 1968). In this scenario, early mortality is not the result of biological damage during development or genetic susceptibility to disease. It is instead the result of an individual having the misfortune to be alive during an extended period of resource scarcity.

A major problem with this potential mechanism of mortality is the time lapse between enamel defect formation and age at death. Enamel defects form during early childhood, with enamel formation complete by age 7 years, except in the third molar (Ten Cate, 1986). The mean age at death for individuals with enamel defects of any kind is 26.92 years, while the mode occurs in the 15–20 year age class. While it is possible for a period of resource scarcity to last for 10–20 years, through gradual population growth coupled with resource depletion, it seems unlikely that a population which relied primarily on hunting and gathering would remain at the same site for that length of time, if this were the case. All archaeological evidence points to abundant and diverse resources at the Libben site. It therefore seems unlikely (though not impossible) that this mechanism played a role at Libben.

CONCLUSIONS

The early mortality of individuals with enamel defects at Libben appears to be most likely due to biological damage to the immune system during development. This damage may occur through prenatal activation of the physiological stress response, iron deficiency during early childhood, or both. Social differences in access to resources, genetic variation in disease susceptibility, and diachronic variation in resource availability were probably not primary causes of early mortality at Libben. The incomplete nature of the archaeological record, however, makes it impossible to rule out any of the latter three mechanisms as secondary causes of early mortality.

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LITERATURE CITED

Acheson RM (1960) Effects of nutrition and disease on human growth. In JM Turner (ed.): Human Growth. New York: Pergamon Press, pp. 73–92.

Baume LJ, and Meyer J (1966) Dental dysplasia related to malnutrition, with special reference to malanodontia and odontoclasia. J. Dent. Res. *45*:726–741.

Bhat MN, and Nelson KB (1989) Developmental enamel defects in primary teeth in children with cerebral palsy, mental retardation, or hearing defects: A review. Adv. Dent. Res. *3*:132–142.

Claman HN (1988) Corticosteroids and the immune response. In GP Chrousos, DL Lauriaux, and PW Gold (eds.): Mechanisms of Physical and Emotional Stress. New York: Plenum Press, pp. 203–208.

Clark GA, Hall NR, Armelagos GJ, Borkan GA, Panjabi MM, and Wetzel FT (1986) Poor growth prior to early childhood: Decreased health and life-span in the adult. Am. J. Phys. Anthropol. *70*:145–160.

Cohen RJ, and Diner H (1970) The significance of developmental enamel defects in neurological diagnosis. Pediatrics *46*:737–747.

Cook DC, and Buikstra JE (1979) Health and differential survival in prehistoric populations: Prenatal dental defects. Am. J. Phys. Anthropol. *51*:649–664.

Falconer DS (1972) Introduction to Quantitative Genetics. New York: The Ronald Press Company.

Federation Dentaire International (1982) An epidemiological index of development defects of dental enamel (DDE Index). Int. Dent. J. *32*:159–167.

Goodman AH, and Armelagos GJ (1985) Factors affecting the distribution of enamel hypoplasias within the human permanent dentition. Am. J. Phys. Anthropol. *68*:479–493.

Goodman AH, and Armelagos GJ (1988) Clinical stress and decreased longevity in a prehistoric population. Am. Anthropol. *90*:936–944.

Goodman AH, and Rose JC (1990) Asessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. Yearb. Phys. Anthropol. *33*:59–110.

Goodman AH, Armelagos GJ, and Rose JC (1980) Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. Hum. Biol. *52*:515–528.

Goodman AH, Thomas R, Swedlund A, and Armelagos GJ (1988) Biocultural perspectives on stress in prehistoric, historic, and comtemporary population research. Yearb. Phys. Anthropol. *31*:169–202.

Grahnen H, and Edlund K (1967) Maternal diabetes and changes in the hard tissues of primary teeth. Odontol. Rev. *18*:157–162.

Grahnen H, Sjolin S, and Magnusson BO (1969) Neonatal asphyxia and mineralization defects of the primary teeth. Caries Res. *3*:301–307.

Guagliardo MF (1982) Tooth crown size differences between age groups: A possible new indicator of stress in skeletal samples. Am. J. Phys. Anthropol. *58*:383–389.

Harrison ML (1978) Taphonomy of the Libben Site, Ottawa County, Ohio. Manuscript of a paper presented at the Annual Meetings of the American Association of Physical Anthropologists, Toronto, Canada.

Kochanowski BA, and Sherman AR (1985) Cellular growth in iron deficient rats: Effect of pre and postweaning iron repletion. J. Nutr. *115*:270–287.

Kreshover SJ (1942) The histopathology of the incisor teeth of mice with experimentally produced tuberculosis. J. Dent. Res. *21*:27–34.

Kreshover SJ (1960) Metabolic disturbances in tooth formation. Ann. N.Y. Acad. Sci. *85*:161–167.

Kreshover SJ, and Clough OW (1953) Prenatal influences on tooth development: Artificially induced fever in rats. J. Dent. Res. *32*:565–577.

Kreshover SJ, Clough OW, and Bear DM (1953) Prenatal influences on tooth development. I. Alloxan diabetes in rats. J. Dent. Res. *32*:246–261.

Kreshover SJ, Clough OW, and Hancock JA (1954) Vaccinia infection in pregnant rabbits and its effect on maternal and fetal dental tissues. J. Am. Dent. Assoc. *49*:549–562.

Lallo J, Armelagos GJ, and Rose JC (1978) Paleoepidemiology of infectious disease in the Dickson Mounds population. Med. Coll. Virginia Q. *14*:12–23.

Lindemann G (1958) Forekomsten af emaljehypoplasi hos born, som har lidt af mavetarmsygdomme. Odontol. Tidskr. *66*:101.

Lovejoy CO, Meindl RS, Przybeck TR, Barton TS, Heiple KG, and Kotting D (1977) Paleodemography of the Libeen Site, Ottawa County, Ohio. Science *198*:291–293.

McCance KL (1990) Stress and disease. In: KL McCance and SE Huether (eds.): Pathophysiology: The Biologic Basis for Disease in Adults and Children. St. Louis: Mosby Yearbook Publ. pp. 279–291.

Mensforth RP, Lovejoy CO, Lallo JW, and Armelagos GJ (1978) The role of constitutional factors, diet, and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. Med. Anthropol. *2*:1–59.

Nikiforuk G, and Fraser D (1981) The etiology of enamel hypoplasia: A unifying concept. J. Pediatr. *98*:888–893.

Noren JG (1984) Microscopic study of enamel defects in deciduous teeth of infants of diabetic mothers. Acta Odontol. Scand. *42*:153–156.

Park EA (1964) The imprinting of nutritional disturbances on the growing bone. Pediatrics *33*:815–862.

Pindborg JJ (1970) Pathology of the Dental Hard Tissues. Copenhagen: Munksgaard.

Pindborg JJ (1982) Aetiology of development enamel defects not related to fluorosis. Int. Dent. J. *32*:123–134.

Rattner LJ, and Meyers HM (1962) Occurrence of enamel hypoplasia in children with allergies. J. Dent. Res. *41*:646–649.

Romain WF (1979) The Libben site, Ottawa county, Ohio. Part II: The subsistence-settlement system. Ohio Archaeol. *29*:42–43.

Romain WF (1980) The Libben site, Ottawa county, Ohio. Part III: Social behavior and organization. Ohio Archaeol. *30*:53–55.

Rosenzweig KA, and Sahar M (1962) Enamel hypoplasia and dental caries in the primary dentition of prematuri. Br. Dent. J. *113*:279–280.

Sawyer DR, and Nwoku AL (1985) Malnutrition and the oral health of children in Ogbomosho, Nigeria. J. Dent. Child. *132*:141–145.

Schusterman S, and Fellers F (1969) The prevalence of enamel defects in childhood nephrotic syndrome. J. Dent. Child. *36*:435–440.

Scrimshaw NS, Taylor CE, and Gordon JE (1968) Interactions of Nutrition and Infection. WHO Monograph 57.

Selye H (1956) The Stress of Life. New York: McGraw-Hill.

Selye H (1971) The evaluation of the stress concept—stress and cardiovascular disease. In L Levi (ed.): Society, Stress, and Disease. Vol. I. London: Oxford University Press.

Sherman AR, and Helyar L (1988) Iron deficiency, immunity, and disease resistance in early life. In: RK Chandra (ed.): Nutrition and Immunology. New York: Alan R. Liss, Inc., pp. 169–195.

Simpson SW, Hutchinson DL, and Larsen CS (1990) Coping with stress: Tooth size, dental defects, and age-at-death. In CS Larsen (ed.): The Archaeology of Mission Santa Catalina de Guale: 2 Biocultural Interpretations of a Population in Transition. Anthropol. Pap. Am. Mus. Nat. Hist. *68*:66–77.

Smith DM, and Miller J (1979) Gastroenteritis, coelic disease, and enamel hypoplasia. Br. Dent. J. *147*:91–95.

Suckling G (1980) Defects of enamel in sheep resulting from trauma during tooth development. J. Dent. Res. *59*:1541–1548.

Suckling G, and Thurley DC (1984) Developmental defects of enamel: Factors influencing their macroscopic appearance. In RW Fearnhead and S Suga (eds.): Tooth Enamel IV. New York: Elsevier Science Publishers, pp. 357–362.

Suckling G, Elliott DC, and Thurley DC (1986) The macroscopic appearance and associated histological changes in the enamel organ of hypoplastic lesions of sheep incisor teeth resulting from induced parasitism. Arch. Oral Biol. *31*:427–439.

Taylor AN, Branch BJ, Van Zuylen JE, and Redei E

(1988) Maternal alcohol consumption and stress responsiveness in offspring. In GP Chrousos, DL Lauriaux, and PW Gold (eds.): Mechanisms of Physical and Emotional Stress. New York: Plenum Press, pp. 311–318.

Ten Cate AR (1986) Tooth eruption. In SN Bhaskar (ed.): Orban's Oral Histology and Embryology. St. Louis: C.V. Mosby Company, pp. 361–374.

Via WF, and Churchill JA (1959) Relationship of enamel hypoplasia to abnormal events of gestation and birth. J. Am. Dent. Assoc. *59:*702–707.

Watson A, Massler M, and Perlstein M (1964) Tooth ring analysis in cerebral palsy. Am. J. Dent. *107:*370–384.

Weichsel ME (1977) The therapeutic use of glucocorticoid hormones in the perinatal period: Potential neurological hazards. Ann. Neurol. *2:*364–366.

White TD (1978) Early hominid enamel hypoplasia. Am. J. Phys. Anthropol. *49:*79–83.

Wolbach JB (1947) Vitamin A deficiency and excess in relation to skeletal growth. J. Bone Joint Surg. *29:*171.